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Case Report

A rare food poisoning: *Clostridium botulinum*

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ABSTRACT

Botulismus is a gram-positive anaerobic, spore-forming bacterium commonly found in soil. Exotaxins secreted by *Clostridium bolulinum* bacteria are heat-labile and irreversibly block the release of acetylcholine at the cholinergic ends of presynaptic and autonomic nerves at the neuromuscular junction after ingestion with food. The incubation period of botulismus is between 18-38 hours, and the first finding is blurred vision and diplopia symptoms due to the involvement of the eye muscles. The most important point in its treatment is early diagnosis and rapid administration of antitoxin. In this article, a case who was successfully treated in our intensive care unit with the diagnosis of botulismus poisoning is presented.

Keywords: Food poisoning, botulism, clostridium

INTRODUCTION

Botulism is a disease caused by exotoxins secreted by *Clostridium botulinum*; a gram-positive anaerobic, spore-forming bacterium commonly found in soil. It is rare but can cause neuroparalysis in skeletal and respiratory muscles at various levels in humans and animals, respiratory failure, and death in severe cases.[1,2] They secrete seven different toxins (neurotoxins A–G) with different serologic properties but similar pharmacologic effects. In humans, toxins A, B, E, and F cause poisoning.[3,4] With technological developments, especially canned vegetables made at home are the cause of botulism rather than ready-made canned food.[5] These neurotoxins, which are the most lethal toxins known, are not resistant to heat and irreversibly block the release of acetylcholine at the presynaptic and autonomic nerves' cholinergic endings at the neuromuscular junction after ingestion with food. The incubation period of botulism is between 18-38 hours, and the first finding is blurred vision and diplopia symptoms due to the involvement of the eye muscles. In the clinical evaluation of the patients, tendon reflexes are decreased, and sensory deficits are in the background. Autonomic cholinergic symptoms such as fatigue, dry mouth, blurred vision, mydriasis, heart rate and blood pressure changes (bradycardia, hypotension), unexpected color changes in the skin, sweating disorders, urinary retention, abdominal pain, nausea, vomiting, and constipation may be observed.[1,3] Toxins can be detected in serum and feces within 3 days after the ingestion of food. Production of bacteria in stool culture after the third day after exposure is the most sensitive method for diagnosis. Guillain-Barré syndrome, myasthenia gravis, poliomyelitis, drug reactions, and other chemical poisons should be considered in the differential diagnosis of botulism. Electromyography (EMG) is useful in the differential diagnosis of other diseases related to botulism.[6,7] The most important point in treatment is early diagnosis and rapid administration of antitoxin. A polyvalent antitoxin is used in treatment. In this report, we aim to present the clinical and electrophysiologic findings of a patient who developed botulism three days after consuming canned frozen peas.

CASE

A 33-year-old woman with no known history of any disease presented to the emergency department with nausea, vomiting, and abdominal pain for two days. No significant pathology was found as a result of the tests performed, and the patient was given symptomatic treatment and discharged. On the third day, the patient was readmitted to the emergency room with complaints of respiratory distress, dysphagia, and hoarseness. No pathology is detected in the evaluation performed by otorhinolaryngology and pulmonology specialists. No pathology was detected in the patient's investigations and cranial imaging. In the first neurological examination, consciousness was clear and cooperative, eye movements were limited to 4 directions, pupils were markedly mydriatic, direct and indirect light reflexes were -, bilateral ptosis was +, swallowing reflex was markedly decreased, speech was hypophonic, 4 extremities were mobile, no side signs were present, deep tendon reflexes were normoactive, and bilateral base skin reflexes were flexor. With the current clinical picture, the patient is hospitalized in the neurology clinic for further investigation and treatment. Electromyography (EMG) is planned for the etiology. One upper and one lower extremity were studied in the EMG, and there were no findings other than low motor CMAP (compound muscle action potential). The patient was transferred to the intensive care unit before the repetitive nerve stimulation test could be performed because the

patient's respiratory failure increased. Within a few hours after transfer, the patient was electively intubated because respiratory failure did not respond adequately to non-invasive methods. Lumbar puncture (LP) was performed to exclude acute polyneuropathy. Cerebrospinal fluid (CSF) examination revealed protein 35.19 mg/dl (reference upper limit 32), glucose 94 mg/dl (concurrent serum glucose 134 mg/dl), and no cells.

Anti-musk (muscle-specific kinase) antibody and acetylcholine receptor antibody tests were sent to the patient to rule out myasthenia gravis (MG). Since myasthenic crises could not be ruled out, a 2-g/kg 5-day IVIG treatment was planned. The LP results and normal deep tendon reflexes ruled us out Miller Fisher's syndrome, which is a variant of Guillain-Barré syndrome with ophthalmoplegia, areflexia, and ataxia. A detailed anamnesis was obtained from the patient's relatives again for botulism disease, which was in the patient's differential diagnosis. When the patient's relative stated that the patient had consumed frozen peas 3 days before the onset of these complaints and since the clinical picture was compatible, a serum sample was taken for toxin type determination and pentavalent botulism antidote was administered. No deterioration in consciousness or significant limb weakness was observed during the daily follow-up in the intensive care unit. A few days later, a decrease in ophthalmoplegia and a decrease in the need for ventilator support were observed.

The antibody results sent to rule out MG were negative, and the serum sample was positive for toxin a and b. The diagnosis of botulism was confirmed. The patient received mechanical ventilator support for one week and then was extubated, and was followed up in the clinic for rehabilitation. The patient whose clinical condition improved in the follow-up was discharged with cure.

DISCUSSION

Clostridium botulinum is a gram-positive bacillus and anaerobic bacterium that can be found widely in the external environment, especially in soil. Botulism is known to be caused by the ingestion of canned foods prepared from foods such as homemade vegetables, meat, fish, and cheese under inappropriate conditions.[1,8,9] While bacterial spores are resistant to high temperatures and can be destroyed by boiling for 2-3 hours, the toxin is heat sensitive and denatures by boiling for 10 minutes.[1] It can cause disease in humans and animals with different strains and different clinical presentations. The main factor causing the disease is the neurotoxin produced by the bacteria. Neurotoxins are classified into seven types, ranging from A to G. In humans, the most common causative agents are A, B, and E. However, neurotoxins F, G, and H may also be the causative agents of the disease, albeit rarely.[10] In our case, both toxin types A and B were positive, as seen in some cases. Symptoms appear 18-38 hours after the ingestion of infected food. In some patients, this period may shorten by up to 2 hours, while in others it may extend by up to 8 days.[1,11] In our case, symptoms appeared within the first 36 hours. Since the disease first affects the ocular and oropharyngeal muscles, the first findings include dry mouth, ptosis, diplopia, limitation in extraocular movements, strabismus, nystagmus, mydriasis, and nonreactive pupils. The triad consisting of extraocular muscle paralysis, pupillary dysfunction, and ptosis is considered being an indicator that the disease is severe and respiratory failure may develop.[1,12] In our patient, complaints of strong swallowing, hoarseness of voice, and visual findings, including difficulty in focusing near, diplopia, and blurred vision, started initially. In the following days, the disease progresses downward and spreads to the extremities and respiratory muscles. Most cases have signs of autonomic dysfunction, such as mydriasis, poor pupillary response to light and distance, dry mouth, constipation, and urinary retention. Mental functions and sensations remain normal.[1,13,14] Serum electrolytes, renal and liver function tests, complete blood and urine tests, electrocardiography (ECG), and cerebrospinal fluid may be completely normal in the absence of secondary complications. No abnormality was found in our patient as a result of these tests, and ECG and biochemistry tests were also found to be normal. The diagnosis of the disease is made by demonstration of botulinum toxin in the blood, demonstration of toxin and/or microorganism in stool or gastric contents, and demonstration of toxin or organism in suspected food.[15] Since botulism is a very rare disease, it is a clinical condition that is very difficult to diagnose, especially if it is not included in the differential diagnosis.[8,10,14] The diagnosis can be confirmed by observation of typical symptoms and EMG findings together with the patient's anamnesis of canned food consumption and the exclusion of other diseases with similar clinical presentations. Although EMG is non-specific, the most common pathologies are low motor compound muscle action potentials (MCAP), inhibition responses in repetitive nerve stimulation, and conduction blocks in single-fiber EMG(Table 1).[16]

Table 1. Demonstration of CMAP amplitude decrease in all four motor nerves studied in our patient

Motor Nerve Conduction						
	Starting (ms)	Time (ms)	Amplitude (mV)	Area (microVs)	Distance (cm)	Speed (m/s)
Right median						
1 Wrist	2.9	12.7	2.5	8.0		
2 Elbow	6.9	15.4	2.4	7.7	23.0	57.5
Right ulnar						
1 Wrist	2.7	13.0	2.7	8.8		
2 Elbow	8.3	8.7	2.8	6.8	25.0	54.6
Right peroneal						
1 Wrist	3.1	8.6	2.4	5.5		
2 Fibula head	9.9	14.5	2.4	9.3	31.2	45.9
Right tibial						
1 Wrist	2.9	12.8	2.5	5.6		
2 Pop. fossa	11.0	12.1	1.9	6.1	41.0	50.6

ETHICAL DECLARATIONS

Informed Consent: All patients signed and free and informed consent form.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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